



UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE
United States Patent and Trademark Office
Address: COMMISSIONER FOR PATENTS
P.O. Box 1450
Alexandria, Virginia 22313-1450
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/798,896	03/11/2004	Eric D. Rabinovsky	AVSI-0034	7397

89065 7590 08/17/2010
VGX Pharmaceuticals, LLC
1787 Sentry Parkway West
Building 18, Suite 400
Blue Bell, PA 19422

EXAMINER

TON, THAIAN N

ART UNIT	PAPER NUMBER
----------	--------------

1632

NOTIFICATION DATE	DELIVERY MODE
-------------------	---------------

08/17/2010

ELECTRONIC

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Notice of the Office communication was sent electronically on above-indicated "Notification Date" to the following e-mail address(es):

US.Patents@inovio.com

Office Action Summary	Application No. 10/798,896	Applicant(s) RABINOVSKY ET AL.	
	Examiner Thaian N. Ton	Art Unit 1632	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on 06 July 2010.
- 2a) ☒ This action is **FINAL**. 2b) ☐ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 17, 21-24, 26, 28, 29, 31, 33, 38, 41 and 42 is/are pending in the application.
- 4a) Of the above claim(s) _____ is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 17, 21-24, 26, 28, 29, 31, 33, 38, 41 and 42 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some * c) ☐ None of:
- ☐ Certified copies of the priority documents have been received.
 - ☐ Certified copies of the priority documents have been received in Application No. _____.
 - ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- | | |
|---|---|
| 1) <input type="checkbox"/> Notice of References Cited (PTO-892) | 4) <input type="checkbox"/> Interview Summary (PTO-413) |
| 2) <input type="checkbox"/> Notice of Draftperson's Patent Drawing Review (PTO-948) | Paper No(s)/Mail Date. _____ |
| 3) <input type="checkbox"/> Information Disclosure Statement(s) (PTO/SB/08) | 5) <input type="checkbox"/> Notice of Informal Patent Application |
| Paper No(s)/Mail Date _____ | 6) <input type="checkbox"/> Other: _____ |

DETAILED ACTION

Applicants' Response, filed 7/6/10, has been entered. Claims 17, 21-24, 26, 28, 29, 31, 33, 38, 41 and 42 are pending and under current examination.

Election/Restrictions

Applicant's election of claims 17-38 (group II), SEQ ID NO:1 and stimulating angiogenesis as the goal of the claimed treatment method in the response on 2/2/2006 is acknowledged. Because Applicant did not distinctly and specifically point out the supposed errors in the restriction requirement, the election has been treated as an election without traverse (MPEP § 818.03(a)).

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

Claims 17, 21, 24, 26, 28, 29, 31, 33, 38, 41, 42 stand rejected under 35 U.S.C. 103(a) as being unpatentable over Alila *et al.* (cited above) in view of Draghia-Akli (cited previously), Fewell *et al.* (cited previously), Gonçalves (Cardiovascular Res., 45: 294-302, 2000), Nicosia *et al.* (American J. of Pathology, 145(5): 1023-1029, 1994) and Isner (cited previously).

Applicants' Arguments. Applicants argue that a prior art reference must be considered in its entirety, *i.e.*, as a whole, including portions that would lead away from the claimed invention. Applicants argue that the combination of two patents, where one of the patents specifically teaches that such combination should not be made, is manifestly improper.

Applicants argue that the Office fails to consider the entirety of the Alila reference, which includes the difficulty and unpredictability of IGF-I therapy for neuronal development due to ineffective expression. Applicants argue that in Alila, of the two products tested *in vivo* in rats, one plasmid (pIG0100) was determined not to be effective, which was explained to be the result of inefficient secretion, and that in addition to this teaching away from the present invention, Alila does not disclose teachings regarding electroporation or the formulation and promoter used in the claimed invention. Applicants argue that considering the unpredictability of the art, as attested to in Alila, the specific teaching away from the present invention, one of ordinary skill in the art would have not had reasonable success in view of Alila to practice the claimed invention. Applicants argue that the secondary references fail to make up for the lack of teaching in Alila, and teach elements that are simply picked and chosen using the instant specification as a roadmap, and thus is impermissible hindsight prohibited by patent laws. See pages 5-6 of the Response.

Response to Arguments. In response to applicant's argument that the examiner's conclusion of obviousness is based upon improper hindsight reasoning, it must be recognized that any judgment on obviousness is in a sense necessarily a

reconstruction based upon hindsight reasoning. But so long as it takes into account only knowledge which was within the level of ordinary skill at the time the claimed invention was made, and does not include knowledge gleaned only from the applicant's disclosure, such a reconstruction is proper. See *In re McLaughlin*, 443 F.2d 1392, 170 USPQ 209 (CCPA 1971).

Regarding Applicants' arguments that Alila teaches away from the claimed invention, the Examiner responds that a teaching away constitutes that a disclosure must criticize, discredit or otherwise discourage the solution claimed (see *In re Fulton*, 391 F.3d 1195, 73 USPQ2d 1141 (Fed. Cir. 2004)). This is not analogous with the instant case. Alila teaches the intramuscular injection of hIGF-I plasmid that produces a localized and sustained level of biologically active hIGF-I (see Abstract). Thus, Alila teaches that hIGF-I can be expressed in muscle tissue. Thus, Alila provides guidance to show that hIGF-I can be used in intramuscular gene therapy. There is nothing in Alila that criticizes, discredits or discourages using hIGF-I in gene therapy. Obviousness does not require absolute predictability, only a reasonable expectation of success, i.e., a reasonable expectation of obtaining similar properties. See, e.g., *In re O'Farrell*, 853 F.2d 894, 903, 7 USPQ2d 1673, 1681 (Fed. Cir. 1988). In the instant case, Alila teaches that a single intramuscular injection of the plasmids resulted in expression for 28 days. They teach that injection of pIG0100, but not pIG0552, resulted in accumulation of intracellular hIGF-I, but that using either plasmid, human hIGF-I mRNA was detectable (p. 1792, col. 1, 1st full ¶). The section to which Applicants refer to (p. 1793, 1st col; 1st ¶) relates to the increase in intracellular accumulation of hIGF-I using pIG0100, but does not teach that using pIG0552 does not result in expression of hIGF-I, as evidenced on the previous page. Alila does not teach away from the claimed invention, because there is nothing in Alila that discourages or specifically discredits or criticizes using plasmids in human IGF-I intramuscular gene therapy. On the contrary, Alila show that using both plasmids, expression of human IGF-I

occurred. Applicants' arguments regarding Alila's lack of teachings the specific promoter used or electroporation or formulation (p. 6, 1st ¶ of the Response) are addressed by secondary pieces of art in the rejection below. Accordingly, the rejection is maintained.

Rejection

Alila *et al.* teach the construction of a plasmid (pIG0552), which contains the 5' portion of the chicken skeletal α -actin gene enhancer/promoter, which is operably linked to the human IGF-I cDNA, and flanked by the 3' portion of human growth hormone UTR (see page 1786, 1st col., 1st ¶ and Figure 1). They teach the purified plasmid was formulated with a complex with PVP (polyvinylpyrrolidone) and then intramuscularly injected into the hind limb of rats (see p. 1787, 1st col., Animal Injections). The muscle samples were then harvested and frozen at various time points and analyzed for hIGF-I expression. Alila *et al.* teach that hIGF-I expression was found localized in the injected muscles (see p. 1790, col. 1-2, bridging ¶). Alila teach intramuscular injection of a construct with a myogenic promoter (chicken skeletal α -actin), which is operably linked to a nucleic acid sequence encoding IGF-I, operably linked to a 3'UTR region, and they teach the expression of this plasmid construct localized to muscle tissue. They teach the limitations of claim 42 because Alila teach the human growth hormone 3'UTR. Alila *et al.* further teach specific embodiments of the claims in that they teach delivery via a single administration (claim 31); delivery into muscle which are diploid cells (claim 33); and that the subject is an animal (rat) (claim 38).

However, Alila *et al.* does not teach a synthetic myogenic promoter that comprises SEQ ID NO:3 (i.e., the synthetic myogenic promoter termed SPc5-12) (claims 17 and 41), nor do they teach a nucleic acid construct comprising an amino acid sequence of SEQ ID NO: 4 (claim 24), or an expression construct that comprises SEQ ID NO: 1 (claim 26) and Alila does not teach transfection enhancing

techniques/compounds such as electroporation or transfection facilitating polypeptides as a means to deliver nucleic acids to cells (claims 17, 28, 29).

However, prior to the time of the claimed invention, Draghia-Akli teaches a myogenic promoter consisting of the nucleic acid of SEQ ID NO:3 (i.e. the synthetic myogenic promoter termed SPc5-12). Draghia-Akli teaches a plasmid construct comprising the SPc5-12 promoter operably linked to a nucleic acid encoding human growth hormone releasing hormone (GHRH; page 1182, col. 2, paragr. 3). Draghia-Akli teaches intramuscular injection of said plasmid construct into pigs and then electroporating the injected muscle of said pig to more efficiently deliver said plasmid to the muscle cells (page 1180: col. 1, paragr. 4, line 1 to col. 2, line 10). Thus, Draghia-Akli teaches that said SPc5-12 promoter is a powerful synthetic muscle promoter that drives high level expression of operably linked heterologous nucleic acids in a muscle-specific manner (page 1180, col. 1, lines 1-2).

Fewell teaches intramuscular injection of plasmid DNA complexed with the charge polypeptide poly-L-glutamate into mice followed by electroporation. Fewell teaches that injection of a plasmid comprising a nucleic acid encoding factor IX and that injection of a plasmid comprising a nucleic acid encoding erythropoietin as such (i.e. forming a complex comprising said plasmids and poly-L-glutamate prior to injection) resulted in enhanced expression of said plasmids compared to when said plasmids were injected as saline solution (i.e. when said plasmids were not complexed with poly-L-glutamate). Thus, Fewell teaches that intramuscular injection of plasmid DNA complexed with poly-L-glutamate followed by electroporation results in more efficient transfection of the cells within the injected muscle.

It would have been obvious to an artisan of ordinary skill at the time of the invention to modify the method of Alila *et al.* with a reasonable expectation of success by: 1) interchanging the avian skeletal chicken skeletal α -actin promoter with the strong muscle-specific synthetic SPc5-12 promoter taught by Draghia-Akli,

2) complexing plasmid DNA with poly-L-glutamate prior to intramuscular injection of said plasmid DNA as taught by Fewell and 3) subjecting muscle tissue injected with said plasmid DNA to electroporation as taught by both Draghia-Akli and Fewell with a reasonable expectation of success. An artisan of ordinary skill would have been motivated to modify the method of Alila as such because: 1) Draghia-Akli teaches that the synthetic SPc5-12 promoter drives high level, muscle-specific expression of operably linked nucleic acids, 2) Fewell teaches that complexing plasmid DNA with poly-L-glutamate prior to intramuscular injection and prior to electroporation results in enhanced uptake of said plasmid DNA and 3) both Draghia-Akli and Fewell teach that electroporating muscle after intramuscular injection of plasmid DNA results in enhanced uptake of said plasmid DNA. Increased cellular uptake of plasmid DNA and increased expression of operably linked nucleic acids contained within said plasmid would be advantageous when practicing methods of gene therapy.

Further, it is noted that pAV2001 (i.e. SEQ ID NO:1 of the instant application) is a hybrid plasmid consisting of fragments of the plasmids taught by Alila (citing Coleman) and Draghia-Akli. The specification on page 42, lines 16-19 recites, "An Nco/HindIII fragment of a SIS II plasmid (Coleman et al., 1995), containing the IGF-I cDNA and the skeletal alpha actin 3'UTR, was cloned into the NcoI/KpnI sites of pSP-HV-GHRH (Draghia-Akli et al., 1999) to generate pSP-IGF-I-SK3'UTR (pAV2001 – SEQID No.: 1)." Thus, an artisan of ordinary skill at the time of the invention would have realized with a reasonable expectation of success that the teachings of Alila (citing Coleman) and Draghia-Akli could be combined to generate the plasmid DNA consisting of the nucleic acid sequence of SEQ ID NO:1.

Although neither Alila, Draghia-Akli or Fewell specifically state that IGF-I is an angiogenic factor, Isner teaches a method for stimulating angiogenesis in an ischemic muscle tissue in a human host comprising injecting into said tissue a DNA sequence encoding an angiogenic protein, wherein said DNA sequence comprises a

promoter sequence, wherein the angiogenic protein is selected from a group of angiogenic proteins including insulin-like growth factor (IGF-I; claims 1 and 16; col. 4, lines 8-10, 23). Thus, Isner identifies IGF-I as an angiogenic protein. Additionally, the prior art is replete with teachings to show that IGF-I is an angiogenic factor. Gonçalves teach that state that, “IGF-I has been shown to be an angiogenic growth factor...” See p. 296, col. 2, #3.1, *Insulin Growth Factors*. They further state that IGF-I seems to participate in inflammation-linked angiogenesis and/or tissue repair (p. 299, col. 1, #4.5, *IGFs and reperfusion*). Similarly, Nicosia *et al.* teach that IGF-I can stimulate rat aortic angiogenesis (see, for example, p. 1024, 1st col., 1st full paragraph). They state that, “Our findings corroborate recent reports that have implicated IGF-I in angiogenesis.” See p. 1027, col. 1-2, bridging sentence. Accordingly, one of skill in the art, given the teachings of both Gonçalves and Nicosia would have had a reasonable expectation of success that utilizing a vector containing IGF-I would stimulate or promote angiogenesis.

Accordingly, in view of the combined teachings, it would have been obvious for one of skill in the art to utilize the methods of Alila, to intramuscularly inject a construct that comprises the construct as taught by Alila, Coleman and Draghia-Akli, and to modify this technique by electroporating the muscle after injection of the plasmid DNA, by methods taught by Fewell, with a reasonable expectation of success. One of ordinary skill in the art would have been motivated to make these modifications, as shown above, that Draghia-Akli teach a strong, muscle-specific promoter, and that complexing plasmid DNA with poly-L glutamate prior to intramuscular injection and electroporation after injection results in more efficient transfection of the cells within the injected muscle. The teachings of Isner, Gonçalves and Nicosia provide additional motivation for an artisan of ordinary skill to use a nucleic acid encoding IGF-I to stimulate angiogenesis in muscle and further support that the claimed invention as a whole was *prima facie* obvious.

Claims 22-23 stand rejected under 35 U.S.C. 103(a) as being unpatentable over Alila *et al.* (cited above) in view of Draghia-Akli (cited previously), Fewell *et al.* (cited previously), Gonçalves (**Cardiovascular Res.**, 45: 294-302, 2000, Nicosia *et al.* (**American J. of Pathology**, 145(5): 1023-1029, 1994) and Isner (cited previously) as applied to claims 17, 21, 24, 26, 28, 29, 31, 33, 38, 41, 42 above, and further in view of van Deutekom *et al.* (Mol. Med. Today, 214-220, May 1998).

Applicants' Arguments. Applicants argue that the Office attempts to create an obviousness argument based on van Deutekom's purported teachings that intramuscular injection of non-viral vectors – such as plasmid DNAs- which are encompassed by the claims, are shown to have low transfection efficiency, and that these efficiencies can be improved by using non-targeted liposomes and/or polylysine-condensed plasmid DNA. Applicants argue that one of ordinary skill would have known that non-viral vectors have a number of options for enhancing transfection efficiency in addition to liposome facilitation, including electroporation, gene gun, gold-assisted deliver, among others. Furthermore, these tools for enhancing transfection are yet to be proven the successful methodology, and thus, one of ordinary skill would not have had reasonable success to practice the claimed invention based on the combination of references. See pages 6-7 of the Response.

Response to Arguments. These arguments have been considered but are not persuasive. It is noted that KSR forecloses the argument that a specific teaching, suggestion, or motivation is required to support a finding of obviousness. See the recent Board decision *Ex parte Smith*, --USPQ2d--, slip op. at 20, (Bd. Pat. App. & Interf. June 25, 2007) (citing KSR, 82 USPQ2d at 1396) (available at <http://www.uspto.gov/web/offices/dcom/bpai/prec/fd071925.pdf>).

The combination of prior art cited above in all rejections under 35 U.S.C. 103 satisfies the factual inquiries as set forth in *Graham v. John Deere Co.*, 383 U.S. 1, 148 USPQ 459 (1966). Once this has been accomplished the holdings in KSR can be applied (*KSR International Co. v. Teleflex Inc. (KSR)*, 550 USPQ2d 1385 (2007):

“Exemplary rationales that may support a conclusion of obviousness include: (A) Combining prior art elements according to known methods to yield predictable results; (B) Simple substitution of one known element for another to obtain predictable results; (C) Use of known technique to improve similar devices (methods, or products) in the same way; (D) Applying a known technique to a known device (method, or product) ready for improvement to yield predictable results; (E) “Obvious to try” – choosing from a finite number of identified, predictable solutions, with a reasonable expectation of success; (F) Known work in one field of endeavor may prompt variations of it for use in either the same field or a different one based on design incentives or other market forces if the variations are predictable to one of ordinary skill in the art; (G) Some teaching, suggestion, or motivation in the prior art that would have led one of ordinary skill to modify the prior art reference or to combine prior art reference teachings to arrive at the claimed invention.”

In the instant case, at least rationales C), D), E) and G) are applicable to the instant situation. In particular, one of skill in the art would have recognized the low transfection efficiency in using plasmid DNA, as taught by van Deutekom. One of skill in the art would have recognized that because of the low transfection efficiency, using non-targeted liposomes and/or polylysine-condensed plasmid DNA could be used to optimize transfection efficiencies. Additionally, one of skill in the art would have a finite number of identified, predictable methods, such as those stated by Applicants (electroporation, gene gun, gold assisted delivery) that would be “obvious to try” any of these, including those taught by van Deutekom, to provide the reasonable expectation of success of transfection. Finally, one of skill in the art would have been motivated to make such a modification, as van Deutekom discuss the low transfection efficiency in intramuscular gene delivery, and suggest using non-targeted liposomes to improve efficiency. See also, MPEP §2143.01 which states, in part: The court stated that “the prior art's mere disclosure of more than

one alternative does not constitute a teaching away from any of these alternatives because such disclosure does not criticize, discredit, or otherwise discourage the solution claimed.." *In re Fulton*, 391 F.3d 1195, 73 USPQ2d 1141 (Fed. Cir. 2004). In the instant case, Applicants have readily identified various techniques that were known to the skilled artisan, thus, one of skill in the art would have known of all these techniques, and utilizing non-targeted liposomes and/or polylysine-condensed plasmid DNA would be obvious in order to improve transfection efficiency. The rejection is maintained.

Rejection

Alila *et al.*, Draghia-Akli, Fewell, Gonçalves, Nicosia and Isner are summarized above. They do not specifically teach mixing the isolated nucleic acid expression construct with a transfection facilitating system before delivery (claim 22); or that the transfection facilitating system is a liposome or cationic lipid (claim 23). However, prior to the time of the claimed invention, van Deutekom teach that intramuscular injection of non-viral vectors – such as plasmid DNAs – which are encompassed by the instant claims, are shown to have low transfection efficiency, and that these efficiencies can be improved by using non-targeted liposomes and/or polylysine-condensed plasmid DNA (see p. 215, 1st col., 1st ¶, Non-Viral Vectors).

Accordingly, given the combined teachings of Alila *et al.* Draghia-Akli, Fewell, Gonçalves, Nicosia and Isner and van Deutekom, it would have been obvious for one of ordinary skill in the art to modify the method of Alila *et al.*, utilizing a modified vector, as suggested by Draghia-Akli, and utilizing electroporation techniques, taught by Fewell, to mix the isolated nucleic acid expression construct with a transfection-facilitation system, such as utilizing a liposome, as contemplated by van Deutekom, with a reasonable expectation of success. Additionally, one of skill in the art would have had a reasonable expectation of success of stimulating angiogenesis, in view of the teachings of Gonçalves, Nicosia and Isner. One of ordinary skill in the art would have been

Art Unit: 1632

motivated to make such a modification, as van Deutekom discuss the low transfection efficiency in intramuscular gene delivery, and suggest using non-targeted liposomes to improve efficiency.

Thus, the claimed invention, as a whole, is clearly *prima facie* obvious in the absence of evidence to the contrary.

Conclusion

No claim is allowed.

THIS ACTION IS MADE FINAL. Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire **THREE MONTHS** from the mailing date of this action. In the event a first reply is filed within **TWO MONTHS** of the mailing date of this final action and the advisory action is not mailed until after the end of the **THREE-MONTH** shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than **SIX MONTHS** from the mailing date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Thaian N. Ton whose telephone number is (571)272-0736. The examiner can normally be reached on 9-5:30 M-F.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Peter Paras can be reached on 571-272-4517. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Thaian N. Ton/
Primary Examiner, Art Unit 1632